

**UNITED STATES DISTRICT COURT
DISTRICT OF NEW JERSEY
CAMDEN VICINAGE**

IN RE	:	
PAULSBORO DERAILMENT CASES	:	MASTER DOCKET NO.:
	:	1:13-CV-784 (RBK/KMW)
	:	
	:	

MICHELLE TRULUCK, et al.	:	NO. 13-CV-5763-RBK-KMW
Plaintiffs,	:	
	:	
v.	:	
	:	
CONSOLIDATED RAIL	:	
CORPORATION, et al.	:	
Defendants	:	
	:	

**PLAINTIFFS' BRIEF IN OPPOSITION TO DEFENDANTS'
MOTION TO EXCLUDE THE EXPERT REPORT AND
TESTIMONY OF OMOWUNMI OSINUBI, M.D.**

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Plaintiffs, Michelle Truluck and Abdeslam Sahla hereby file their joint response to the Motions the Exclude the Reports and Testimony of Dr. Omowunmi Osinubi filed by defendants Consolidated Rail Corporation, Norfolk Southern Railway Company and CSX Transportation.

INTRODUCTION

On November 30, 2012, a train derailed while crossing the Paulsboro Moveable Bridge as a result of the tortious conduct of Defendants Consolidated Rail Corporation, Norfolk Southern Railway Company, and CSX Transportation, Inc. (collectively, “Defendants”), releasing well over 20,000 gallons of vinyl chloride gas into the air. The small town was blanketed in a carcinogenic haze within a matter of hours.

Plaintiffs, Michelle Truluck and Abdeslam Sahla were among the many who were exposed to vinyl chloride and its equally toxic breakdown products. Both suffered symptoms of acute exposure, and both have experienced more chronic, long-term health effects as well.

Truluck and Sahla have each proffered expert reports authored by Dr. Omowunmi Osinubi in their respective cases. Her reports include opinions regarding their exposures and the injuries that were caused by the toxic release, as well as recommended programs of medical monitoring tailored to their individual situations.

Defendants have moved to exclude all testimony of Dr. Osinubi, generally as a part of a larger tactical effort to strike all expert testimony proffered by the plaintiffs in this litigation and also, purportedly, on a litany of alleged defects in her methodology and analysis. As discussed below, however, the defendants have failed to prove that Dr. Osinubi's testimony will be deficient under either Fed.R.Evid. 702 or *Daubert*, and their Motions to exclude her reports and/or testimony should be denied.

BACKGROUND

I. The Derailment Resulted in a Massive Exposure to Vinyl Chloride.

Raelynn Stevenson was drinking her morning cup of coffee at her home when she witnessed the derailment. She described a vapor cloud that appeared right after the train cars derailed that looked like "the dust that came up the street that you saw on TV on 9/11. That's what it looked like coming up to my house. It looked like the 9/11 dust." *See* Declaration of David Cedar ("Cedar Dec.") Exhibit A (NTSB Interview of Raelynn Stevenson ("R. Stevenson Statement")), at p. 9:15-6; 10:6-16. Before the accident, the weather "was crystal clear. It was a beautiful morning." R. Stevenson Statement, p. 11:17-8

Conrail's engineer confirmed that "as soon as the top of the bridge went down it was almost instantaneously that the fog bank came up out of the Mantua

Creek.” *See* Cedar Dec. Exhibit D (Deposition Transcript of Mark Mather (“Mather Dep.”)), p. 103:12-14. Conrail’s engineer described the size of the vapor cloud: “Pretty much the whole neighborhood . . . had a fog.” *Id.* at 113:19-21.

The official Paulsboro police report stated: “as [Patrolman Rodney Richards] was speaking with [Conrail’s] conductor, [he] noticed a smoky fog start to swarm the immediate area and become very thick. The smoky substance that quickly surrounded [him] caused a reaction that made [him] cough several times.” Cedar Dec. , Exhibit K (Paulsboro Police Report), p. 2.

The recorded communications of the first responders further supports the immediate presence of toxic fog:

7:01 “It’s [the train] is spewing out all kinds of gas.” (Gloucester County 911 call)

7:05 “Rail cars or tank cars have been pierced and have leaked out all of their contents into the creek. The creek is full of vapors from these cars.” (Channel 3 fire Ops.)

7:06 “It’s a major emergency, bridge collapsed and major hazards, potentially life-threatening...I have an odor out here that they are not familiar with. This odor is hazardous. Hazard released.” (Zone 3 Police Radio Channel)

See Cedar Dec. Exhibit N (Timeline of Events and Communications on November 30, 2012).

The ruptured tank car contained about 25,000 gallons, or about 177,000 pounds, of vinyl chloride. The National Oceanic and Atmospheric Administration (NOAA) ran an Area Locations of Hazardous Atmospheres (“ALOHA”) model of

what the exposures would be if the entire amount were released through a hole 12 inches in diameter over a time period of two minutes. This model showed that the “toxic threat zone” would extend out two miles from the release area. *See Cedar Dec. Exhibit P (NOAA ALOHA Model for Paulsboro, NJ).* The toxic threat zones are assessed in terms of Acute Exposure Guideline Levels (“AEGLs”) established by the National Advisory Committee managed by the Environmental Protection Agency.

Severity Tier	Vinyl Chloride Air Concentration (ppm)			Definition
	Exposure for 10 minutes	Exposure for 30 minutes	Exposure for 60 minutes	
AEGL-1	450	310	250	The airborne concentration of a substance above which it is predicted that the general population, including susceptible individuals... ...could experience notable discomfort, irritation, or certain asymptomatic nonsensory effects. However, the effects are not disabling and are transient and reversible upon cessation of exposure.
AEGL-2	2,800	1,600	1,200	...could experience irreversible or other serious, long-lasting adverse health effects or an impaired ability to escape.
AEGL-3	12,000	6,800	4,800	...could experience life-threatening health effects or death.

See Cedar Dec., Exhibit T (NJ DOH Air Quality Consultation).

The highest threat zone, corresponding to AEGL-3 at 4,800 ppm, would extend 1,383 yards, or about 0.8 mile, from the spill site; the next highest threat zone, corresponding to AEGL-2 at 1,200 ppm, would extend from 0.8 to 1.2 miles

away from the spill site; and the last threat zone, corresponding to AEGL-1 at 250 ppm, would extend from 1.2 to 2.0 miles away from the spill site. *See* Cedar Dec. Exhibit P. An Interagency Modeling and Atmospheric Assessment Center (“IMAAC”) model similarly estimated that the AEGL-3 zone would be about 0.5 mile in diameter and the total toxic threat zone about 1.0 mile in diameter. *See* Cedar Dec. Exhibit Q (IMAAC Model).

Conrail’s contractor determined that when turned to gas, the entire 80,000 kg of vinyl chloride would spread over a surface area of 30,000 square meters and one meter thick. *See* Cedar Dec. Exhibit R (ARCADIS US’s Vinyl Chloride Model). This means that an area encompassing approximately three square blocks of Paulsboro would be 100% vinyl chloride, or, in the alternative, that there would be enough vinyl chloride to contaminate an area of 300 city blocks to a level of 10,000 parts per million if equally disbursed.

II. Paulsboro Refinery Vinyl Chloride Testing Results

Paulsboro Refinery personnel arrived at the scene of the derailment at about 8:30 a.m. with Photo Ionization Detectors (“PID”). They found that the levels of vinyl chloride in the air were so high that they were unable to “zero” out their equipment. Without being able to “zero” out the PIDs first, the readings on these devices yielded results of 631, 694 and 760 ppm. As analytic chemical expert, Dr. Brian Buckley, explains, these readings need to be multiplied by 1.9 to obtain a

vinyl chloride equivalent. See Cedar Dec. Exhibit S (Report of Brian Buckley (“Buckley Report”)), p. 1.

About ten minutes later, the Paulsboro Refinery employees walked a few blocks away to Delaware Street and Billings Avenue to try to zero out their instruments, but instead obtained readings well in excess of 100 ppm. *Id.* at 2. The same meters later on had negative readings ranging from -40 to -60 ppm which is, of course, impossible. *Id.* Dr. Brian Buckley reviewed the Paulsboro Refinery data and opined that the actual vinyl chloride levels present in the air at the time the Paulsboro Refinery readings were taken were “greatly in excess of the readings recorded by the device.” *Id.* at 3. Due to being unable to properly zero the device out and to the excessive saturation of the chemical, the readings from the Paulsboro Refinery PIDs could reasonably be expected to result in much lower numbers than was actually present. *Id.*

III. New Jersey Department of Health Consultations

The New Jersey Department of Health (“DOH”) performed a “Health Consultation” on air quality in Paulsboro following the derailment. The DOH analyzed the air modeling data that was done and concluded that “[b]ased on modeled estimates and monitoring, peak air concentrations . . . exceeded the EPA’s Acute Exposure Guidance Levels (AEGL) for one hour exposure that are associated with reversible health effects (AEGL-1: 250 ppm) and possibly

disabling effects (AEGL-2: 1,200 ppm) or life threatening effects (AEGL-3: 4,800 ppm).” Cedar Dec. Ex. I at 11.

V. Odor Detection and Threshold

Many in Paulsboro complained of the sickly sweet odor of vinyl chloride on the day of the chemical spill. One of the complainants, Stephanie Esposito, a Fox 29 news reporter, tweeted at 9:13 a.m. on November 30th that she “just took a walk down to the roadblock and definitely smell[ed] something sweet.” She described the odor as “a pungent smell [that] hits you like a brick wall when you walk into it”. She was able to smell this odor even though she had a cold. *See* Cedar Dec. Exhibit Y, Screenshot of Stephanie Esposito’s Twitter Feed. Ptl. Richards, who had worked in Paulsboro since 2006, testified that the odor of chemical was distinct from the typical smells in the town. He too described it as a “pungent smell”. *See* Cedar Dec. Exhibit L (Deposition Transcript of Rodney Richards (“Richards Dep.”)), p. 31-32.

Dr. Maria Kent, who is a family practitioner in Paulsboro, testified that multiple patients were concerned about their vinyl chloride exposure and reported complaints of nausea, headaches, wheezing, confusion, dizziness and dry throat. *See* Cedar Dec. Exhibit AA (Deposition Transcript of Dr. Maria Kent (“Kent Dep.”)) pp. 41-43. A number of these patients also complained of smelling some kind of chemical odor that day. *Id.*

The Agency for Toxic Substances and Disease Registry (“ATSDR”) also reports the odor threshold for vinyl chloride to be “about 3,000 ppm” noting that it varies significantly among individuals. *See* Cedar Dec. Exhibit AC (ATSDR Report on Vinyl Chloride).¹ An odor threshold test performed by Union Carbide on a panel of experts with an average of ten years experience in odor detection and evaluation found that the odor threshold for vinyl chloride was commonly 2,000 ppm, although two of the most sensitive panelists could detect a faint odor at 1,200 ppm. *See* Cedar Dec. Exhibit AD (Union Carbide Corporation Vinyl Chloride Odor Threshold Test).

The New Jersey Department of Health took two surveys of residents of Paulsboro following the train wreck. The first was an in person “door to door” survey and the second was a mailed survey. 50% of the adults in the in-person survey reported smelling or tasting unusual odors; in the mailed survey 69% of households reported that at least one member of the household smelled or tasted an unusual odor. *See* Cedar Dec. Exhibit AE (NJ DOH Health Consultation), p. 8. Among those who reported smelling or tasting an odor in both the in-person and mailed surveys, there were higher frequencies of reported new or worsening symptoms. Also in both surveys, the most frequently reported symptoms among those who smelled an odor were headache, coughing, irritation of the nose and

¹ The NJDOH Hazardous Substance Fact Sheet states “Odor threshold = > 3,000 ppm.”

throat, dizziness, irritation or pain or burning of eyes, and difficulty breathing. *Id.*

The DOH concluded that:

The symptoms commonly reported are consistent with what is known to occur from exposure to vinyl chloride, specifically headache, irritation of the eyes, nose, throat and lungs, coughing, nausea, and dizziness or lightheadedness. ... [T]here was a similar pattern of reported symptom frequencies between the in person and mailed surveys, as well as with the findings of surveys of emergency responders.

Id. at 12.

The report also found that “symptoms were most commonly reported from evacuated areas and areas within one block of evacuated areas and were least frequent in areas farther than 3,500 feet from the derailment location.” *Id.*

VI. Vinyl Chloride Health Information

The New Jersey DOH has a hazardous substance fact sheet for vinyl chloride, which states:

Vinyl chloride is a CARCINOGEN in humans. There may be no safe level of exposure to a carcinogen.

The acute health effects include the following:

- Exposure to **Vinyl Chloride** can severely irritate and burn the skin and eyes with possible eye damage. Contact with the *liquid or gas* can cause frostbite.
- Inhaling **Vinyl Chloride** can irritate the nose, throat and lungs causing coughing, wheezing and/or shortness of breath.
- **Vinyl Chloride** can cause headache, nausea, vomiting, dizziness, fatigues, weakness and confusion. Higher levels can cause lightheadedness and passing out.

See Cedar Dec. Exhibit AF, NJ DOH Hazardous Substance Fact Sheet. OSHA's permissible exposure limit for vinyl chloride is 1 ppm averaged over an 8 hour day. The short-term exposure limit is 5 ppm, not to be exceeded during any 15-minute period. *Id.* OSHA recommends medical monitoring consisting of liver function tests, chest X-rays and lung function tests for workers exposed to 0.5 ppm of vinyl chloride. *See* Cedar Dec. Exhibit AG, OSHA Standards 1910.1017.

The International Agency for Research on Cancer's (IARC) Monograph on the Evaluation of Carcinogenic Risk to Humans found sufficient evidence that vinyl chloride causes angiosarcomas of the liver and hepatocellular carcinomas (HCC). *See* Cedar Dec. Exhibit AH (IARC Monograph on Evaluation of Carcinogenic Risk to Humans, Volume 97 (2008)), p. 425. The same report cited numerous studies which "found evidence of a significant association between exposure to vinyl chloride monomer (VCM) and mortality from liver cirrhosis. *Id.* at 327 (citing The European Multicentric Study); *id.* at 328 (citing Pirastu, et al., 2003). Mastrangelo in 2004 also reported "an association between exposure of VCM and both liver cirrhosis and [hepatocellular carcinoma]." *Id.* at 329; *see also id.* at 349 (a Cross Sectional Study of Hepatocellular Carcinoma in Italy found an association between exposure to VCM and both liver cirrhosis and HCC in vinyl chloride workers). IARC concluded "together with the observation that vinyl chloride increases the risk for liver cirrhosis, which is a known risk factor for

hepatocellular carcinoma, these findings provide convincing evidence that vinyl chloride causes hepatocellular carcinoma as well as angiosarcoma of the liver.” *Id.* at 422.

In addition, the Material Safety Data Sheet (MSDS) of Oxyvinyl issued for this very shipment of vinyl chloride risks among potential health effects states the following:

Inhalation: May cause respiratory tract irritation. Several minutes of exposure to high, but attainable concentrations (over 1,000 ppm) may cause difficulty breathing, central nervous system depression and symptoms such as: ataxia or dizziness, drowsiness or fatigue, loss of consciousness, headache, euphoria and irritability, visual or hearing disturbances, nausea, memory loss. Prolonged high concentration exposure may cause unconsciousness or death. Cardia: Acute intoxication may cause irregular heartbeats.

See Cedar Dec. Exhibit AI, MSDS on Vinyl Chloride (Monomer), p. 10.

Mastrangelo, et al.’s 2004 study explicitly concluded that “VCM exposure appears to be an independent risk factor for [hepatocellular carcinoma] and [liver cirrhosis].” *See Cedar Dec. Exhibit AJ, Mastrangelo, et al., Increased Risk of Hepatocellular Carcinoma and Liver Cirrhosis in Vinyl Chloride Workers: Synergistic Effect of Occupational Exposure with Alcohol Intake, Vol. 112 (2004), p. 1192.*

VII. Facts Specific to Michelle Truluck

On the morning of the incident, Michelle Truluck was at work when she was contacted on her phone by her nephew, William Jones (of whom she is the legal guardian) and her niece Cashmere, who told her that due to the train accident, their school was closed, and they were being sent back home. She left work to come home and let them into the house since they didn't have a key. On her way, she was driving with the windows open, but when she arrived back to the area near her home she encountered a "thick fog" which was difficult to even see through – the vapor cloud caused by the released vinyl chloride. Truluck Dep., Exhibit "A" at 22-29. Ms. Truluck resides at 1101 Chestnut Street in Paulsboro, which, according to MapQuest, is 0.16 miles from the site of the derailment. This was well within the "red zone" defined by the initial evacuation area. See Report of Omowunmi Osinubi, Exhibit "B" at 3, 12, 16. See also Report of Panos Georgopoulos, Exhibit "C" at 6, 11.

Based upon the available air modeling data, and given Ms. Truluck's proximity to the derailment site and her reported symptoms as compared to symptoms described in the available scientific literature, Ms. Truluck's exposure to vinyl chloride, particularly on the date of the derailment, likely exceeded AEGL-3 level by a significant margin. She remained in her home, located in the red zone, until the afternoon of Sunday, December 2, 2012. Accordingly, her exposure

window likely exceeded 57 hours in all. Osinubi Report, Exhibit “B” at 16.

Ms. Truluck experienced profound symptoms of acute exposure shortly after coming into contact with the fog. She developed an irritating red, itchy and bumpy skin rash on her face and arms. She also immediately experienced coughing, and severe headaches she described as “unbearable.” Other symptoms she experienced included blurred vision, red, tearing and itchy eyes, nasal irritation, nausea and vomiting as well as diarrhea. Truluck Dep., Ex. “A” at 29, 32- 37 and 47.

By Sunday, two days after the vinyl chloride release, Ms. Truluck’s symptoms had worsened, prompting her to seek treatment at the Emergency Department of Underwood Hospital, where she received a chest x-ray, but was not offered a breathing test. Upon discharge from the E.R., she was advised by hospital staff to evacuate her home. Following her evacuation from her home that day, her respiratory symptoms persisted, including cough and sinus irritation. Osinubi Report at 3-5; Truluck Dep. at 47:10-48:12

On December 11, 2012, almost two weeks after the derailment, Ms. Truluck continued to suffer from the symptoms that began with her exposure. She was seen at her primary physician’s office on that date with complaints of severe chest pain, with a persistent cough and green sputum, facial rash and diarrhea. She related that her symptoms began right after the derailment. She was prescribed a cortisone cream, put on a liquid diet, and instructed on skin care. Diagnosis was

contact and suspected exposure to potential chemical hazards. Osinubi Report at 7; Truluck Dep. at 49:5-24.

Since the time of the derailment and continuing to the present, she has continued to suffer from a persistent cough accompanied by discharge of green mucus. This is associated with chest pain. She has also developed abdominal pain. She has had abnormal breathing tests and has been diagnosed with sleep apnea, which she treats with the use of a CPAP machine. Osinubi report at 3-5. These symptoms are all consistent with symptoms associated with AEGL-2 and AEGL-3 levels of vinyl chloride exposure. The symptoms experienced by Ms. Truluck have been reported by individuals exposed to levels from 12,000 to 25,000 ppm. Osinubi Report at 16.

VIII. Facts Specific to Abdeslam Sahla

On the morning of the derailment, Mr. Sahla drove from his home at 33B Baird Avenue in Paulsboro to the Billingsport School, at 441 Nassau Avenue in Paulsboro, arriving there around 7:20 or 7:25 a.m., so that he could spend time with his children before the school doors opened at 7:45 a.m.. He drove to the school via Delaware Street. At the time, there was a lot of activity with a helicopter circling overhead and a fog in the air; there was also an unusual odor in the air. His children went into the school, Mr. Sahla returned home, and then he

retrieved his children from school shortly after the school called him around noon. Sahla Dep., Exhibit “A” at 27:6-34:9.

Mr. Sahla’s travels on the morning of the derailment and several hours thereafter placed him directly into contact with vinyl chloride in substantial concentrations, particularly in the morning. Additionally, his home was well within the “red zone” defined by initial and subsequent air modeling data, therefore exposing him to vinyl chloride and its breakdown products. See Report of Panos Georgopoulos, Ph.D. (Exhibit “C”) at pp. 3-5 and 11-16 and Report of Omowunmi Osinubi, M.D. (Exhibit “E”) at 3-4, 10-12 and 18-20.

Within a few days of the derailment, Mr. Sahla experienced some symptoms of coughing and shortness of breath. See Answers to Interrogatories (No. 4) (attached to Defendants’ Motion as Exhibit “B”). See also Sahla Dep., Exhibit “D” at 48:1-19.

Since the derailment, Mr. Sahla (who is now 42) experienced progressive difficulty breathing through his nose beginning in 2013. Although he had a remote nasal fracture as a child, his nasal obstruction inexplicably became worse shortly after the derailment, and worsened to the point where he consulted an otolaryngologist (ENT), Dr. David Bromberg in September of 2014. Sahla Dep. at 61:2 – 18, 63:12-64:12. Dr. Bromberg saw evidence of the old injury (described as “twisting” of the septum) but he also saw polypoid changes of the left middle

turbinate. See 9/24/14 office note of David Bromberg, M.D. (Exhibit “E”). Dr. Bromberg felt surgical correction would be necessary, and prescribed a CT of the sinuses to determine if there was evidence of a persistent sinusitis. *Id.* On 9/29/14, CT of the sinuses showed that in addition to the polypoid changes which Dr. Bromberg was able to visualize during clinical evaluation, there were also visible mucosal thickening in both maxillary sinus, and several polyps in the right maxillary sinus. See CT Scan Report dated 9/29/14, attached as Exhibit “F”. Shortly thereafter, on 10/24/14, Dr. Bromberg performed nasal septoplasty and bilateral endoscopic inferior turbinectomy to remove the obstruction. *See* Exhibit “E”.

IX. Injuries and Conditions Causally Related to Vinyl Chloride Release

Per the reports (Exhibits “B” and “G”), deposition testimony (Exhibit “H”) and attached Declaration of Dr. Omowunmi Osinubi (Exhibit “I”), the differential diagnosis for Ms. Truluck’s respiratory problems is vinyl chloride-related reactive airway dysfunction syndrome, or irritant-induced exacerbation of her quiescent asthma/pre-existing hyper-reactive airways, and/or interstitial lung disease. All of these conditions should be attributed to her vinyl chloride exposure. Dr. Osinubi recommends further testing to be done to establish a definitive diagnosis for each of these conditions as are clinically indicated.

The differential diagnoses for Mr. Sahla is vinyl chloride breakdown

product-related irritation of nasal and sinus tissues resulting in mucosal thickening (polypoid changes) of the maxillary sinus and polypoid changes/hypertrophy of the turbinate. *See* Exhibits “G” and “H”.

ARGUMENT

“The Rules of Evidence embody a strong and undeniable preference for admitting any evidence which has the potential for assisting the trier of fact.” *Kannankeril v. Terminix Int’l Inc.*, 128 F.3d 802, 806 (3d Cir. 1997) (citing *Holbrook v. Lykes Bros. S.S. Co.*, 80 F.3d 777, 780 (3d Cir. 1996)); *see also* Fed. R. Evid. 402 (“Relevant evidence is admissible.”). If expert evidence is admissible, the trier of fact will determine the proper weight to give it. *Maloney v. Microsoft Corp.*, 2011 U.S. Dist. LEXIS 127870, at *6-7 (D.N.J. Nov. 4, 2011).

In considering pre-trial challenges to expert testimony, Rule 702 has “three major requirements: (1) the proffered witness must be an expert, *i.e.*, must be qualified; (2) the expert must testify about matters requiring scientific, technical or specialized knowledge; and (3) the expert’s testimony must assist the trier of fact.” *Pineda v. Ford Motor Co.*, 520 F.3d 237, 244 (3d Cir. 2008).² “A district court’s inquiry under Rule 702 is ‘a flexible one’ and must be guided by the facts of the case.” *ZF Meritor, LLC v. Eaton Corp.*, 696 F.3d 254, 294 (3d Cir. 2012).

² Defendants do not challenge Dr. Osinubi’s qualifications.

I. Dr. Osinubi did not rely on the NTSB's report.

Defendants claim that to the extent Dr. Osinubi “relied upon” the NTSB Final Report, her opinions are completely inadmissible under 49 U.S.C.A. § 1554 (b), which provides that “no part of a report of the Board, related to an accident or investigation of an accident, may be admitted into evidence or used in a civil action for damages resulting from a matter mentioned in the report.”

The majority of courts nationally hold, however, that this exclusionary rule prohibits admission *only of NTSB opinions and conclusions*, while allowing admission of factual findings. *See Curry v. Chevron USA*, 779 F.2d 272, 274 (5th Cir.) (citing *Am. Airlines v. United States*, 418 F.2d 180 (5th Cir. 1969)); *Travelers' Ins. Co. v. Riggs*, 671 F.2d 810, 816 (4th Cir. 1982) (noting that 803(8) allows admission of reports generally, but the exclusionary rule forbids admission of at least the conclusory portions of the NTSB reports)); *Chrion Corp. & PerSeptive Biosystems, Inc. v. Nat'l Transp. Safety Bd.*, 198 F.3d 935, 940–41 (D.C. Cir. 1999) (holding that NTSB regulations precluded admission of any part of the NTSB accident report but that investigators' reports are fully admissible notwithstanding the exclusionary rule).

Moreover, it is inaccurate and misleading to claim that Dr. Osinubi “*relied* upon the NTSB Accident Report and Factual Report as the underlying basis for *all* of her opinions in this case.” Defs. Br. at 6 (emphasis added). Although Dr.

Osinubi reviewed the NTSB's Accident Report, she did not "rely" on it to support her substantive opinions. On the contrary, as is evident from her reports, she relied on a mountain of other exposure-levels evidence (cited above), including the Air Quality Consultation of the New Jersey Department of Health and the Air Modeling Report of Dr. Georgopoulos, both of which confirm that the levels were, in fact, very high.

During her deposition, Dr. Osinubi referenced the NTSB, the New Jersey DOH, the EPA, ALOHA modeling, and Dr. Georgopoulos collectively merely to note the universal agreement of credible sources that exposure levels were very high. *See* Osinubi Dep. at 44-47. Her casual reference to the NTSB is inconsequential to the opinions she will offer at trial. Defendants' argument for exclusion under 49 U.S.C. § 1154(b) is thus a red herring and should be summarily rejected.

II. Dr. Osinubi used well-established and generally accepted methodology to opine on general causation.

Dr. Osinubi's general causation methodology is not only logical but generally accepted in her field and the courts. She looked to see if there was a high exposure, and (since there was such an exposure) whether the symptoms experienced by the plaintiff began shortly after the exposure and were consistent with what would be expected to result from it. *See* Osinubi Dec. at ¶ 3. This was the identical methodology used by the New Jersey Department of Health in its own

health consultation for Paulsboro and accepted by Dr. Greenberg (Defendants' expert) himself. *See Id.* at ¶ 5.

A. Dr. Osinubi's general causation opinion easily survives under *Kannankeril v. Terminix Int'l*, 128 F.3d 802 (3d Cir. 1997).

In *Kannankeril*, the plaintiff claimed she had a cognitive impairment caused by exposure to pesticides applied by Terminix. The trial court struck the expert's causation testimony on grounds that there was no air-testing sufficient to support the expert's opinion about the plaintiff's exposure and that the expert's opinion on causation was unreliable and unsupported by fact, the same arguments made by Defendants here. The Third Circuit reversed. In discussing the exposure, the Third Circuit rejected the notion that the plaintiff's expert had to rely on ambient air tests (which were not conducted until 9 months after the application of pesticides), and found it sufficient for the expert to look at Terminix's application records showing when, how much and where pesticide had been applied. *Kannankeril*, 128 F.3d at 808-809.

Critical to the instant motion, the Third Circuit in *Kannankeril* held that “***all factual evidence*** of the presence of the chemicals in the residence should be relevant in forming an expert opinion of causation.” *Id.* at 809 (emphasis added). The Third Circuit's holding cements the principle that *Daubert* reliability determinations must be made upon consideration of the full evidentiary record,

which will dictate whether or not certain exclusionary principles are apt.

In this case, although there are no precise measurements of either plaintiff's exposure (as is typically the case in an environmental release affecting the general public), it is undisputed that 23,000 gallons of vinyl chloride were released into the environment and that this amount of vinyl chloride would fill up a cloud over 27,000 cubic meters in size of 100% vinyl chloride. This is more than analogous to the "application records of how much pesticide was applied" in the *Kannankeril* case. If that were not enough, there is substantial evidence that both plaintiffs drove through a vinyl chloride cloud (indeed, Truluck lived in extremely close proximity to the bridge), began to experience symptoms at or shortly after the time of exposure, and smelled vinyl chloride in the air, which is detected by most people only at levels *substantially* in excess of anything resembling a safe exposure limit. Additionally, there is Dr. Georgopoulos's model and the NJ DOH report, both of which document extensive levels of exposure and demonstrate that both Sahla and Truluck's geographic position on November 30, 2012 subjected them to those high levels.³

³ Because this extensive exposure evidence supports Dr. Osinubi's opinions, there is no need to discuss Defendants' pointless attack on the ALOHA model. *See* Defs. Br. at 12-15. In addition, plaintiffs in the various cases pending respond to the attack on Dr. Georgopoulos's air model in their joint opposition to Defendants' motion (ECF No. 737) to exclude his testimony. It suffices to say here that "when direct measurements cannot be made, exposure can be measured by mathematical modeling, in which one uses a variety of physical factors to estimate the transport

In *Kannankeril*, the expert's methodology was indistinguishable from that employed by Dr. Osinubi here: "The temporal relationship and nature of her complaints led me to conclude that with reasonable medical certainty the cause of Dr. Kannankeril's central nervous system manifestations of toxicity is exposure to Dursban." *Kannankeril, supra*, 128 F.3d at 805. The Third Circuit concluded that because the plaintiff's expert had based his opinion on the plaintiff's medical records and reports of the volume of pesticide applied and his general experience, general medical knowledge, standard text books, and standard references, the experts "opinion on causation has a factual basis and supporting scientific theory."

There is a legion of case-holdings in line with Dr. Osinubi's methodology – that an acute exposure closely followed by symptoms known to result from that exposure provides "good grounds" for an expert's opinion on causation. *See, e.g., Thomas v. CMI Terex Corp.*, 2009 U.S. Dist. LEXIS 86623, at *40 (D.N.J. Sept. 21, 2009) (Simandle, J.) ("The question of causation can be resolved by a doctor without even medical testing, where the temporal proximity between an accident and the subsequent injury make the accident the most probable cause of the injury.") (collecting cases). In *Winnicki v. Bennigan's, et al.*, 2006 U.S. Dist. LEXIS 5568 (D.N.J. Feb. 9, 2006) (Greenaway, Jr.), for example, the court evaluated expert testimony in a case in which the plaintiff ate a Caesar salad the

of the pollutant from the source to the receptor." Reference Manual on Scientific Evidence, p. 424 (2d ed. 2000).

night before she became sick with acute gastrointestinal dysfunction, which led to kidney failure and death. Although the expert could never determine exactly what was wrong with the salad, he opined that the salad was the cause of the condition using a differential diagnosis and temporal relationship. The court denied the defendant's motion to exclude the expert, citing Third Circuit precedent (*e.g.*, *Kannankeril*) accepting "medical testimony that relies heavily on a temporal relationship between and illness and a causal event." *Id.* at *46.

B. Causation can be established in the absence of a precise measurement of plaintiffs' levels of exposure, particularly where, as here, there is abundant evidence of substantial exposure.

The only distinction between the defense expert's (Dr. Greenberg) methodology and that of Dr. Osinubi is that Dr. Greenberg would require idiosyncratic measurement of exposure to conclude that there is a completed exposure pathway. *Cf. id.* at ¶¶ 4, 6. But that was not required by the New Jersey DOH, nor is it required by the pertinent case law.⁴ As the Third Circuit explained in *Heller v. Shaw Indus., Inc.*, 167 F.3d 146, 157 (3d Cir. 1999), "even absent hard evidence of the level of exposure to the chemical in question, a medical expert could offer an opinion that the chemical caused plaintiff's illness." As another court

⁴ Of course, record evidence already shows that, according to the New Jersey DOH, "[i]n Paulsboro there *was* a completed exposure pathway to Vinyl Chloride in the hours and days following the derailment" Osinubi Dec. at ¶ 7 (quoting NJ DOH Air Quality Health Consultation, p. 6) (emphasis modified).

has explained, in rejecting a railroad's *Daubert* challenge:

We disagree with CSX that in order to validate the testimony of the medical experts, Moody was required to prove the precise dosage of solvents to which he was exposed and the precise level required to have a harmful effect on human beings. * * *

We believe that Moody presented sufficient evidence both as to his level of exposure and that necessary to cause his toxic encephalopathy. He presented testimony concerning how often he used the offending solvents and the duration of his exposure. He further explained the physical symptoms that he suffered while working with the solvents. While not quantitatively specific, the expert testimony supports the conclusion that Moody's exposure, under the circumstances described, and his length of the exposure, are sufficient to cause his toxic encephalopathy.

CSX Transp., Inc. v. Moody, 2007 Ky. App. LEXIS 208, at *18-19 (Ky. Ct. App. July 13, 2007); see also *Whitlock v. Pepsi Ams.*, 527 Fed. App'x 660, 661-662 (9th Cir. 2013) ("Plaintiffs' **probable** ingestion of TCE-contaminated groundwater," coupled with the fact "that the alleged TCE and chromium exposure levels were 'within [a] reasonable range of that known [from several studies] to induce' the alleged injuries" was sufficient for expert testimony to satisfy *Daubert*; "Whether [that testimony] proves causation is not a question of admissibility.") (emphasis added); *Louderback v. Orkin Exterminating Company*, 26 F. Supp. 2d 1298, 1306-07 (D. Kan. Oct. 14, 1998) (as long as expert considered facts of plaintiff's exposure, the temporal relationship between exposure and disease, the plaintiff's medical records and history of disease, then an expert's opinion on causation is considered reliable and clearly "has a factual basis and supporting scientific

theory” even when there is no specific evidence of exposures in excess of the ACGIH threshold level or the EPA reference dose); *Harris v. Peridot*, 313 N.J. Super 257, 298 (N.J. Super. Ct. - App. Div. 1998) (holding that an expert could reasonably consider the fact that the injuries sustained are consistent with a high level of exposure on “both sides of the equation”, i.e. as additional evidence supporting the conclusion that the exposure was substantial).

C. The Bradford Hill factors are not a *per se* requirement, and do not apply in cases focusing on acute exposure.

While Defendants focus almost exclusively on the “Bradford Hill” methodology, that approach does not govern a case of acute exposure causing acute injury. *See* Osinubi Dec. at ¶ 1. As noted, *supra*, courts recognize that a strong temporal relationship coupled with immediate symptomatology can support a conclusion of causation. *See, e.g., In re Stand ‘N Seal Prods. Liab. Litig.*, 623 F. Supp. 2d. 1355, 1371-72 (N.D. Ga 2009) (causation opinion that exposure caused chemical pneumonitis survived *Daubert* challenge because strong temporal relationship between exposure and acute onset of respiratory symptoms, despite lack of dose/response data); *In re Ephedra Prods. Liab. Litig.*, 2007 U.S. Dist. LEXIS 74914, at *7 (S.D.N.Y. Oct. 5, 2007), *vacated and remanded on other grounds by Giordano v. Market Am., Inc.*, 289 Fed. App’x 467, 469 (2d Cir. 2008) (“The close temporal proximity between Ms. Stafford’s stroke and her use of ephedra, coupled with the general-causation evidence about ephedra’s rapidly

acting biological effects (in contrast to asbestos), permit a jury to infer that the dose she ingested was sufficient to be considered a substantial factor in causing her stroke.”); *see also Cavallo v. Star Enter.*, 892 F. Supp. 756, 774 (E.D. Va. 1995) (“[T]here may be instances where the temporal connection between exposure to a given chemical and subsequent injury is so compelling as to dispense with the need for reliance on standard methods of toxicology.”); *accord Nat’l Bank of Commerce v. Dow Chemical Co.*, 965 F. Supp. 1490, 1525 (E.D. Ark. 1996).

Sir Bradford Hill himself recognized that his viewpoints could be irrelevant in the case of acute exposure to indisputably toxic chemicals. *See* Osinubi Dec. at ¶ 1 (“A particular and perhaps extreme physical environment cannot fail to be harmful. A particular chemical is known to be toxic to man therefore suspect on the factory floor. Sometimes alternatively, we may be able to consider what might a particular environment do to a man ***and then see whether such consequences are, in deed, to be found.***”) ; *see also Milward v. Acuity Specialty Prods. Group*, 639 F.3d 11, 17 (1st Cir. 2011) (“None of my nine viewpoints can bring indisputable evidence for or against the cause-and-effect hypothesis and none can be required as a *sine qua non*.”) (quoting Austin Bradford Hill); Federal Judicial Center, *Reference Manual on Scientific Evidence* 600 (3d ed. 2011) (“There is no formula or algorithm that can be used to assess whether a causal inference is appropriate based on these guidelines. One or more factors may be absent even

when a true causal relationship exists.”). Dr. Osinubi, for her part, acknowledged the Bradford Hill viewpoints and provided a sound basis for her alternate methodology.⁵

D. Vinyl chloride odor thresholds provide an acceptable basis for assessing exposure .

Defendants’ argument that “[o]dor threshold is not a valid and reliable technique for determining” chemical exposure, Defs. Br. at 12 (citing only their own expert) is false.⁶ Courts routinely recognize that odor threshold is admissible as evidence of exposure when actual measurements are not available. *See, e.g., Taylor v. Union Pacific Railroad Co.* 2010 U.S. District LEXIS 96802, at *12-12, 24-26 (S.D. Ill. Sept. 16, 2010) (holding that experts could conclude the exposure to sulfuric acid in excess of OSHA limits occurred because the odor threshold was at least the OSHA limit and multiple workers could smell the odor); *BP Amoco v. Flint Hills Res.*, 2009 U.S. Dist. LEXIS 131282, at *16 (N.D. Ill. June 3, 2009) (odor threshold testimony deemed non-speculative and admissible); *Magistrini v. One Hour Martinizing Dry Cleaning*, 180 F. Supp. 2d 584, 614 (D.N.J. 2002)

⁵ Defendants’ invocation of the latinism “*post hoc, ergo propter hoc*,” *see* Def. Br. at 10, is unavailing. Dr. Osinubi does not base her opinion that plaintiffs’ exposure to vinyl chloride was substantial solely as a result of their symptomology; she bases it on evidence that both were situated in an area pummeled by mass quantities of vinyl chloride gas at the time when concentrations were highest.

⁶ It is also disingenuous insofar as at least one of the Defendants has taken the exact opposite position in prior litigation. *Cf. Sunnycal v. Csx Transp., Inc.*, 926 F. Supp. 2d 988, 998 (S.D. Ohio 2013) (“CSX argues that Dr. Green should have been allowed to testify about the odor threshold of ‘chlorine gas’ . . .”).

(same). *Roney v. Gencorp*, 2009 US Dist. LEXIS 85816 (S.D.W.V.) (denying *Daubert* challenge of expert who relied on odor threshold to estimate vinyl chloride exposure); *Lewis v. Airco*, 2011 NJ Unpublished LEXIS 191400 at *20 (App. Div.) (recognizing odor threshold as relevant to estimate exposure to vinyl chloride).

While Dr. Osinubi acknowledged a potential deficiency in using odor threshold with respect to a single, unique ‘smeller,’ she *also* testified that “most people” would smell the chemical if the exposure were high enough. *See* Osinubi Dep. at 88-89. And the exposure levels here *were* high enough, because a sizable portion of the surveyed population reported smelling and tasting unusual odors on the day of the derailment. *See* NJ DOH Health Consultation, p. 8.

E. Dr. Osinubi’s review of the New Jersey DOH survey does not diminish the reliability of any of her opinions.

The DOH survey results are plainly admissible as public reports under Fed. R. Evid. 803(8), and any challenge to the methodology used by the NJ DOH goes only to the *weight* the survey evidence should be ascribed. *Cf. In re Nautilus Motor Tanker Co.*, 85 F.3d 105, 113 (3d Cir. 1996) (“[P]ublic reports are presumed admissible in the first instance and the party opposing their introduction bears the burden of coming forward with enough ‘negative factors’ to persuade a court that a report should not be admitted.”); *Ellis v. Int’l Playtex, Inc.*, 745 F.2d 292, 303 (4th Cir. 1984) (“Playtex’s concern about the methodology of the studies should have

been addressed to the relative weight accorded the evidence and not its admissibility.”). Defendants speculate that the survey evidence was “biased,” but that is hardly a sound basis for excluding a public survey. *Cf. Ellis*, 745 F.2d at 303 (“[A]llegations of bias are purely speculative. All epidemiological studies that might implicate a manufactured product are conducted with the possibility of litigation on the horizon.”).

Moreover, Defendants mischaracterize the evidence by saying the survey was “self administered” when, in fact, there were two surveys, and the door-to-door survey variety had results very similar to the mailed survey and was not self administered. Nor is the result from the survey “counterintuitive” because “in some instances reported symptoms increased as the distance from the derailment site increased.” As Dr. Osinubi explained in deposition, decay products are a potential cause of the symptoms complained of, but they may not form until the vinyl chloride traveled some distance from the immediate area. Osinubi dep. at 130-132. In any event, the overall pattern strongly shows symptoms were much more prevalent in people living within 3,500 feet of the derailment than those who live further than 3,500 feet and certainly further than 4,500 feet. *Id.*

F. The absence of studies or literature assessing identical high-level, non-occupational acute exposure to vinyl chloride monomer does not at all render Dr. Osinubi's general causation opinion inadmissible.

There is no requirement “that a medical expert must always cite published studies on general causation in order to reliably conclude that a particular object caused a particular illness.” *Heller v. Shaw Indus.*, 167 F.3d 146, 155 (3d Cir. 1999); *accord Kudabeck v. Kroger Co.*, 338 F.3d 856, 862 (8th Cir. 2003). That precedent—which by itself should permit the Court to skip Defendants’ argument at pages 17-18 of their brief—was applied in *Best v. Lowe's Home Ctrs., Inc.*, 563 F.3d 171 (6th Cir. 2009), where the Sixth Circuit reversed exclusion of plaintiff’s medical expert, Dr. Moreno. The court stated:

Based on his medical knowledge, Dr. Moreno compiled a list of possible causes for the injury, including virus, accident, brain tumor, brain surgery, exposure to chemicals, medications, or an ideopathic (unknown) cause. Lowe's strongest argument is that no published material confirms that inhalation of the chemical in Aqua EZ can cause anosmia. But ‘there is no requirement that a medical expert must always cite published studies on general causation in order to reliably conclude that a particular object caused a particular illness.’ Dr. Moreno did not arbitrarily ‘rule in’ Aqua EZ as a potential cause, but instead concluded from the MSDS sheet and his own knowledge of medicine and chemistry that the chemical it contains can cause damage to the nasal and sinus mucosa upon inhalation.

Id. at 180-181 (internal citation omitted).

Evidence of a chemical’s properties and known effects can be reliably

applied to novel settings in the absence of medical literature directly on point. *See* Osinubi Dec. at ¶ 42 (“Defendants criticize my citation to studies of World Trade Center victims, or occupational exposures because they are ‘dissimilar.’ But there is no large compendium of studies of effects of a 23,000 gallon release of vinyl chloride into a community of 6,000 people. Under these circumstances physicians look to analogous conditions.”).⁷

Notwithstanding this, Dr. Osniubi has supplied a massive amount of medical literature to support her conclusions, which can be seen in her reports and Declaration.

G. Causation of Respiratory Disease

It is undisputed that vinyl chloride is a respiratory irritant, and that its decay products, HCL and formaldehyde, are even more irritating. *See, e.g.*, Osinubi Dec. at ¶¶ 20-22. And there is no question that exposure to a high level of Vinyl Chloride or its decay products, hydrochloric acid and formaldehyde, can cause respiratory problems. *See Id.*

The New Jersey Department of Health Hazardous Substance Fact Sheet for Vinyl Chloride states “Inhaling Vinyl Chloride can irritate the nose, throat and

⁷ Quantitative and qualitative concerns about medical literature go the weight, not admissibility, in any event. *Cf. McCulloch v. H.B. Fuller Co.*, 61 F.3d 1038, 1042 (2d Cir. 1995) (holding that peer review and publication or general acceptance of an expert's theory goes to the weight of the testimony rather than its admissibility).

lungs,” *see id.* at ¶ 12, and Dr. Greenberg agrees that Vinyl Chloride is a respiratory irritant. *See id.* (citing Greenberg Dep. at 26:14-16).

Dr. Osinubi cites a wealth of peer reviewed literature to support her opinion that exposure to respiratory irritants such as vinyl chloride “can result in long term respiratory sequelae, such as asthma, reactive airway dysfunction syndrome (“RADS”), bronchiolitis obliterans or constrictive bronchiolitis and vocal cord dysfunction. RADS is associated with a vapor, mist, fume or gas exposure that is extremely high level and brief, and causes people to develop permanent respiratory problems. *See* Osinubi Dec. at ¶ 15 (citing Greenberg Dep. at 197:13-19); *see also id.* (“Constrictive Bronchiolitis may occur where there is inhalation of gases, toxic fumes, or irritants such as Nitrogen Dioxide, Chlorine Gas, and Mustard Gas.”) (quoting *Small airway disease related to occupational exposures* Gulati, et al. and Clinical Pulmonary Medicine Volume 22, No.3, May 2013, Pg. 133).

A respected textbook in occupational medicine recognizes that “inhalational injury from high intensity exposures” such as those that occur in “transportation accidents” can have serious respiratory effects including irritation, inflammation, pneumonitis, asthma, pulmonary fibrosis and bronchiolitis obliterans.” *Id.* at 18-19. The same textbook confirms that “irritant induced asthma occurs ... after substantial exposure to an irritating dust, mist, vapor or fume.” *Id.* at 20. A more recent article ascribes the emerging spectrum bronchiolar disorders from

occupational and environmental exposures to a wide range of respiratory irritants.

Osinubi Dec. ¶ 30.

Hydrochloric Acid and Formaldehyde are both strongly irritating to the respiratory tract and associated with a wide range of adverse respiratory effects:

Vinyl Chloride Decay Toxicants	Health Effects
Hydrochloric acid ⁸	<ul style="list-style-type: none"> • Severe irritation of respiratory tract, characterized by coughing, choking, or shortness of breath. Severe over-exposure can result in death. • Inflammation of the eye is characterized by redness, watering, and itching. • Skin inflammation is characterized by itching, scaling, reddening, or, occasionally blistering. • Repeated or prolonged exposure to spray mist may produce respiratory tract irritation leading to frequent attacks of bronchial infection.
Formaldehyde ⁹	<ul style="list-style-type: none"> • Corrosive: It causes skin irritation which may range from mild to severe with possible burns, brownish discoloration of the skin, urticaria, pustulovesicular eruptions, irritant and/or allergic dermatitis (eczema). • May be absorbed through the skin with symptoms paralleling those of ingestion. • Causes irritation of the respiratory tract (nose, throat, airways). Symptoms may include dry and sore mouth and throat, thirst, and sleep disturbances, difficulty breathing, shortness of breath, coughing, sneezing, wheezing rhinitis, chest tightness, pulmonary edema, bronchitis, tracheitis, laryngospasm, pneumonia, and palpitations.

⁸ Source: Hydrochloric Acid Material Safety Data Sheet.
<http://www.sciencelab.com/msds.php?msdsId=9924285>

⁹ Source: Formaldehyde Acid Material Safety Data Sheet.
<http://www.sciencelab.com/msds.php?msdsId=9924095>

Vinyl Chloride Decay Toxicants	Health Effects
	<ul style="list-style-type: none"> • Increased risk of asthma and/or allergy observed in humans breathing 0.1 to 0.5 ppm • Eczema and changes in lung function observed at 0.6 to 1.9 ppm • Central nervous system (CNS) effects include: excitement, CNS depression, somnolence, convulsions, stupor, aggression, headache, weakness, dizziness, drowsiness, and/or coma. • Causes gastrointestinal irritation with nausea, vomiting (possibly with blood), diarrhea, severe pain in mouth, throat and stomach. • Damages the kidneys, liver, central nervous system • Classified as carcinogenic by IARC, ACGIG, NTP.

Injuries caused by vinyl chloride and its decay products are analogous to injuries caused by relatively low-dose exposures to chlorine, *e.g.* in a swimming pool which generates hydrochloric acid. *See* Osinubi Dec. at ¶ 17 (citing *Short term respiratory effects of acute exposure to chlorine due to a swimming pool accident* OEM 2001; 58 399-404) (recent reports have documented long term effects, such as asthmatic reactions, bronchial hyper responsiveness, and reduced lung function among exposed people both in general and in the work environment).

Dr. Osinubi has good grounds to analogize vinyl chloride to other solvents and respiratory irritants. Members of the medical science community have rightly observed that “[s]ometimes chemicals of a common type cause a generalized adverse response. For example, nearly all organic solvents from petroleum

products . . . share some (but not all) symptoms in common: ‘defatting’ of the skin following dermal exposure, and central nervous system depression . . . following relatively high levels of inhalation exposure.” *In re Stand 'n Seal, supra*, 623 F. Supp. 2d at 1375 (quoting David L. Eaton, *Scientific Judgment and Toxic Torts - A Primer in Toxicology for Judges and Lawyers*, 12 J.L. & Pol’y 5, 10 (2003)).

H. Increased Risk of Cancer Justifying Medical Monitoring

Dr. Osinubi’s opinion that the massive short term exposure to vinyl chloride justifies medical monitoring because of increased cancer risk is supported by a substantial body of scientific evidence:

1. OSHA requires medical monitoring for workers exposed to vinyl chloride and specifically requires any employee who is exposed to a “massive release of vinyl chloride” as a result of a catastrophic mishap “shall be afforded appropriate medical surveillance.” 29 CFR §1910.1017(b)(5) and §1910.1017(k)(3).
2. The National Academy of Sciences has calculated a 1 in 10,000 cancer risk associated with the following short term exposures to vinyl chloride: 1 in 10,000 for a 30 minute exposure of 2,990 ppm or a one hour exposure of 670 ppm; or 1 in 10,000 for a 30 minute exposure of 1,180 ppm or a one hour exposure of 350 ppm. Osinubi Dec. ¶47.
3. In a study of cancer induction following single and multiple exposures to vinyl chloride, a single exposure to a high dose was found to cause cancer in mice, leading the author to conclude “one dose is sufficient if the dose is high enough.” *Cancer Induction Following Single and Multiple Exposures to a Constant Amount of Vinyl Chloride Monomer*, R. M. Hehir, *Environmental Health Perspectives* Vol. 41pgs. 63-72, 1981¹⁰.

¹⁰ Dr. Osinubi’s opinion cannot be excluded simply because she relied on animal studies. *In re Paoli R.R. Yard Pcb Litig.*, 35 F.3d 717, 743 (3d Cir. Pa. 1994)

4. Using an EPA algorithm for assessing excess cancer risk, Dr. Greenberg admitted that a 60 minute exposure at 4,800 ppm would result in an excess cancer risk of 1 in 11,170. At 90 minutes the incidence would be 50% higher or about 1 in 7,000.¹¹

Even more concerning is the recent study of chromosomal aberration in persons exposed to a vinyl chloride train wreck in Germany. Cf. Osinubi Dec. ¶ 48. In that case, the atmospheric concentration of vinyl chloride was not measured until 15 hours after the event, at which time it was 1 – 8 ppm, certainly no higher than what was found in Paulsboro. That study, which controlled for demographic variables and smoking showed those exposed to vinyl chloride had a statistically significantly increase in the mean frequency of aberrant cells. Dr. Osinubi has cited other articles showing “that a single exposure is effective as multiple exposures in producing chromosome damage” and that there is a significant

¹¹ Dr. Greenberg’s initial calculation using the EPA algorithm was one excess cancer in 333 Million, but he later admitted that this was mistaken by a factor of 1,000 and the actual increased cancer risk is one in 333,000. He based this upon an average exposure of 372 ppb, or 0.968 mg/m³ over an 18 day period. See Greenberg Exhibit 12, 6/16/15 and Greenberg 6/16/15 Dep. at 105:5-109:7. Because Dr. Greenberg’s cancer risk assessment did not include any readings taken before the afternoon of November 30, *Id.* at 114:11-22, he was asked to assume that in lieu of the exposures he calculated over the 18 days, there was a single exposure of 4,800 ppm for an hour and asked to calculate an increased cancer risk on that. Using the same EPA algorithm, he concluded the increased cancer risk would be 29.81 times greater than the 1 in 333,000 cancer risk he had calculated earlier, which results in an excess cancer risk of 1 in 11,170. Greenberg Dep. at 114:23-122:25. Dr. Greenberg then admitted that if the exposure assumed was 4,800 ppm for an hour and a half, the cancer risk would go up by 50%, which would in turn result in an excess cancer risk of about 1 in 7,062, well in excess of the 1 in 10,000 standard universally regarded by regulatory authorities as an excess risk of concern. *Id.* at 123:13-18.

correlation between chromosomal aberrations and the incidence of cancer. Osinubi 5/17/15 Report at p. 14. Dr. Osinubi notes that mutant biomarkers have been found even in workers exposed below OSHA's permissible exposure limit of 1 ppm. *Id.* at 14-15. Accordingly, there is a strong scientific basis to show that increased chromosomal aberrations would be expected to increase cancer risk. *See* Osinubi Dep. at 122:6-10 ("Chromosomal aberrations have been demonstrated to be predictive of cancer development in workers who have been exposed to vinyl chloride.")

Therefore, for all of these reasons Dr. Osinubi's opinions on disease causation are amply supported in record and should not be excluded.¹²

Medical Tests to Evaluate GERD are Recoverable

Contrary to what Defendants claim, Dr. Osinubi does not opine that vinyl chloride exposure "caused" gastroesophageal reflux disease (GERD), but simply that exposure to respiratory irritants, such as vinyl chloride, formaldehyde and HCL can cause or aggravate irritant induced aerodigestive dysfunction syndrome,. Osinubi Dec. at ¶41. Indeed, an increased incidence of these disorders have been found among Persons exposed to the New York disaster area in the aftermath of the World Trade Center attacks and GERD has been specifically named as a

¹² Defendants' attack on Dr. Osinubi's opinions regarding sleep disorder has no place in this case, as Dr. Osinubi did not include these conditions within the differential diagnosis of Mr. Morris.

disease for which compensation may be obtained for WTC exposures. 42 USC 300 mm-22 (a)(3)(A)(xi). Vinyl chloride, hydrochloric acid, and formaldehyde were all found in the complex mix of WTC exposures. Osinubi Dec. at 43, 45.

Plaintiffs are entitled to recover not only for injury already inflicted, but also for expenses reasonably necessary to avert further harm. *See* Restatement 2d of Torts § 919(2) (“One who has already suffered injury by the tort of another is entitled to recover for expenditures reasonably made or harm suffered in a reasonable effort to avert further harm.”).¹³ *See also Theobald v. Angelos*, 40 N.J. 295, 304, 191 A.2d 465, 470 (1963) (“reasonable compensation” includes all expenses reasonably necessary or incidental to plaintiff’s efforts to cure or alleviate his injuries).

III. Defendants lodge no credible challenge to Dr. Osinubi’s differential diagnosis. Her opinion on specific causation is plainly admissible.

The reliability of differential diagnosis has been approved in this circuit. *Heller, supra*, 167 F.3d at 154-155 (3rd Cir 1999); *Paoli III*, 35 F.3d 717, 742, n.8 (3rd Cir. 1994). “To properly perform a differential diagnosis, an expert must perform two steps: (1) ‘Rule in’ all possible causes of [injury] and (2) ‘Rule out’

¹³ The first illustration to Section 919(2) is directly applicable here. “A negligently hits and bruises B’s leg. B applies a dressing to the wound but, reasonably believing that the bone in his leg may be fractured, has X-ray photographs taken. These reveal no fracture. B is entitled to recover the expense of the X-ray photographs.” Restatement 2d of Torts § 919(b), cmt. b, illustration 1.

causes through a process of elimination whereby the last remaining potential cause is deemed the most likely cause of [injury].” *Feit v. Great W. Life & Annuity Ins. Co.*, 271 Fed. App’x 246, 254 (3d Cir. 2008). Furthermore, expert testimony on causation is not inadmissible “simply because it fails to account for some particular condition or fact which the adversary considers relevant.” *Creanga v. Jardal*, 185 N.J. 345, 360 (2005).

CONCLUSION

For all of the reasons given above, Defendants motion to exclude the testimony of Dr. Osinubi in this case should be denied.

Respectfully submitted,

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